SUPPLEMENTARY ABSTRACTS

1048 CHANGES IN PLASMA PREKALLIKREIN, HIGH AND LOW MOLECULAR WEIGHT KININOGENS IN DISSEMINATED INTRAVASCULAR COAGULATION

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Changes in components in kallikrein-kinin system have been studied in 15 cases with disseminated intravascular coagulation (DIC). Nine out of the total cases had infection and six cases had malignancy as primary diseases. Plasma prekallikrein (PKK) was determined by a radiochemical assay using ^{3}H-TAME according to Imanari et al.(1974). High and low molecular weight kininogens (HMWK and LMWK) were bioassayed according to Uchida and Katori (in press). If plasma factor XII, measured in each case, was less than 50 % of normal, values of PKK and HMWK in examined plasma were caliculated from those in 1:1 mixture of examined plasma and normal plasma. All of PKK, HMWK and LMWK Showed moderate to marked decrease in every case with DIC. The values for PKK, HMWK and LMWK were 0.23 0.07 TAME unit/ml (normally 0.62 0.13), 0.20 0.07 ug(bradykinin equiv.)/ml (normally 0.66 0.14) and 1.24 0.44 ug/ml (normally 2.57 0.41), respectively. There was a significant correlation between PKK and antithrombin III levels (r=0.66, p 0.001) in those cases with DIC. PKK was also directly correlated with HMWK and factor XII. Decrease in PKK and HMWK may be chiefly due to consumption. The mechanism for reduction in LMWK, however, remains to be determined. It may be concluded that plasma prekallikrein, high and low molecular weight kininogens decrease in DIC.

1049 SNAKE VENOMS AND HAEMOSTASIS: SUGGESTED MECHANISMS OF THEIR ACTION *IN VIVO* AND *IN VITRO*

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Details of the *in vitro* mode of action of the thrombin-like enzymes from four species are described, with emphasis on their ability to release fibrinopeptides A and B from fibrinogen and their activation of Factor XIII, thus promoting fibrin crosslinking. It was found that *Bitis gabonica* venom removed both A and B peptides from fibrinogen and thus may be considered most similar to thrombin; this venom also causes the activation of Factor XIII, as does thrombin; *Trimeresurus erythrusus* and *T.popeorum* venoms released only fibrinopeptide A, as does *Agkistrodon rhodostoma* and *Bothrops atrox*. The partly purified procoagulant fractions used in these studies activated Factor XIII in the following ascending order: *A.rhodostoma*, *B.atrox*, *T.erythrusus* and *T.popeorum*.

Classification of fibrinogen and its fragments during controlled infusion of Ancrod indicated that the lysis of soluble, non-crosslinked fibrin polymers by plasmin was the mechanism of defibrination following the introduction of a DIC-like syndrome. *Crotalus atrox* venom dramatically defibrinated and affected a wide variety of clotting parameters during snake bite. A mechanism explaining the DIC-like defibrination syndrome associated with snake bite is presented.